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     $0.02  TELNET
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     $0.29  Estimated total session cost   0.148 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R)  1969-2004/Feb W5
         (c) 2004 BIOSIS
  File  73:EMBASE  1974-2004/Feb W5
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  File 155:MEDLINE(R)  1966-2004/Feb W5
         (c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers
have changed. Please see HELP NEWS 154 for details.
  File 399:CA SEARCH(R)  1967-2004/UD=14010
         (c) 2004 American Chemical Society
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Alert feature enhanced for multiple files, etc. See HELP ALERT.

      Set  Items  Description
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? e au=sackstein robert ?

Ref   Items  Index-term
E1        2  AU=SACKSTEIN R.D.
E2       87  AU=SACKSTEIN ROBERT
E3        0 *AU=SACKSTEIN ROBERT ?
E4        1  AU=SACKSTEIN ROBERT D
E5        2  AU=SACKSTEIN ROBERTO
E6        6  AU=SACKSTEIN, R.
E7       24  AU=SACKSTEIN, ROBERT
E8        1  AU=SACKSTEIN, ROBERTO
E9        2  AU=SACKSTEM R
E10      78  AU=SACKSTON W E
E11       9  AU=SACKSTON, W. E.
E12       1  AU=SACKTON B

          Enter P or PAGE for more
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              0  AU=SACKSTEIN ROBERT ?
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              2  AU=SACKSTEIN ROBERTO
              6  AU=SACKSTEIN, R.
             24  AU=SACKSTEIN, ROBERT
              1  AU=SACKSTEIN, ROBERTO
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            347  HECA
              0  52 OF L
          33059  SELECTIN
              0  HECA(W)52 OF L(W)SELECTIN
        1871655  E
          33059  SELECTIN
          13245  E(W)SELECTIN
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09/619290

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3/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014542076 BIOSIS NO.: 200300497104
Prevention of leukocyte migration to inflamed skin with a novel fluorosugar modifier of cutaneous lymphocyte-associated antigen.
AUTHOR: Dimitroff Charles J (Reprint); Kupper Thomas S; **Sackstein Robert**
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Avenue Louis Pasteur, Room 650, Boston, MA, 02115, USA**USA
AUTHOR E-MAIL ADDRESS: cdimitroff@rics.bwh.harvard.edu
JOURNAL: Journal of Clinical Investigation 112 (7): p1008-1018 October 2003 2003
MEDIUM: print
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

3/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014116452 BIOSIS NO.: 200300075171
Glycosylation-dependent inhibition of cutaneous lymphocyte-associated antigen expression: Implications in modulating lymphocyte migration to skin.
AUTHOR: Dimitroff Charles J; Bernacki Ralph J; **Sackstein Robert** (Reprint
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Ave Louis Pasteur, Room 671, Boston, MA, 02115, USA**USA
AUTHOR E-MAIL ADDRESS: rsackstein@rics.bwh.harvard.edu
JOURNAL: Blood 101 (2): p602-610 January 15, 2003 2003
MEDIUM: print
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

3/3/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013765542 BIOSIS NO.: 200200359053
Direct real-time observation of E- and P-selectin-mediated rolling on cutaneous lymphocyte-associated antigen immobilized on western blots
AUTHOR: Fuhlbrigge Robert C; King Sandra L; Dimitroff Charles J; Kupper Thomas S; **Sackstein Robert** (Reprint
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Avenue Louis Pasteur, Boston, MA, 02115, USA**USA
JOURNAL: Journal of Immunology 168 (11): p5645-5651 June 1, 2002 2002
MEDIUM: print
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

3/3/4 (Item 4 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

0013627075 BIOSIS NO.: 200200220586
Homing and hematopoiesis: HCELL is the principal **E-selectin** and L-selectin ligand of human hematopoietic stem cells
AUTHOR: **Sackstein Robert** (Reprint); Dimitroff Charles J (Reprint); Lee Jack Y (Reprint); Fuhlbrigge Robert C (Reprint); Parmar Kalindi; Mauch Peter M; Sandmaier Brenda M
AUTHOR ADDRESS: Dermatology and Medicine, Brigham and Women's Hospital, Boston, MA, USA**USA
JOURNAL: Blood 98 (11 Part 1): p710a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

3/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013153171 BIOSIS NO.: 200100325010
CD44 is a major **E-selectin** ligand on human hematopoietic progenitor cells
AUTHOR: Dimitroff Charles J; Lee Jack Y; Rafii Shahin; Fuhlbrigge Robert C; **Sackstein Robert** (Reprint
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Ave. Louis Pasteur, Room 671, Boston, MA, 02115, USA**USA
JOURNAL: Journal of Cell Biology 153 (6): p1277-1286 June 11, 2001 2001
MEDIUM: print
ISSN: 0021-9525
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

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           33059  SELECTIN
           13245  E(W)SELECTIN
             347  HECA
          327269  52
               0  HECA(W)52
           15453  SULFATION
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          756101  INDEPENDENT
      S6       7  S5(20N)(INDEPENDENT)
? rd s6
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? t s7/7/all

7/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013953211 BIOSIS NO.: 200200546722
Distinct sulfation requirements of selectins disclosed using cells that support rolling mediated by all three selectins under shear flow. L-selectin prefers carbohydrate 6-sulfation to tyrosine sulfation, whereas P-selectin does not
AUTHOR: Kanamori Akiko; Kojima Naoya; Uchimura Kenji; Muramatsu Takashi; Tamatani Takuya; Berndt Michael C; Kansas Geoffrey S; Kannagi Reiji (Reprint)
AUTHOR ADDRESS: Program of Molecular Pathology, Aichi Cancer Center, Research Inst., Nagoya, 464-8681, Japan**Japan
JOURNAL: Journal of Biological Chemistry 277 (36): p32578-32586 September 6, 2002 2002
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: L- and P-selectin are known to require sulfation in their ligand molecules. We investigated the significance of carbohydrate 6-sulfation and tyrosine sulfation in selectin-mediated cell adhesion. COS-7 cells were genetically engineered to express P-selectin glycoprotein ligand-1 (PSGL-1) or its mutant in various combinations with 6-O-sulfotransferase (6-Sul-T) and/or alpha1 fwdarw 3fucosyl-transferase VII (Fuc-T VII). The cells transfected with PSGL-1, 6-Sul-T, and Fuc-T VII cDNAs supported rolling mediated by all three selectins and provided the best experimental system so far to estimate kinetic parameters in selectin-mediated cell adhesion for all three selectins using the identical rolling substrate and to compare the ligand specificity of each selectin. L-selectin-mediated rolling was drastically impaired if the cells lacked carbohydrate 6-sulfation elaborated by 6-Sul-T, but not affected when PSGL-1 was replaced with a mutant lacking three tyrosine residues at its NH2 terminus. L-selectin-mediated adhesion was also hardly affected by mocarhagin treatment of the cells, which cleaved a short peptide containing sulfated tyrosine residues from PSGL-1. In contrast, P-selectin-mediated rolling was abolished when PSGL-1 was either mutated or cleaved by mocarhagin at its NH2 terminus, whereas the cells expressing PSGL-1 and Fuc-T VII but not 6-Sul-T showed only a modest decrease in P-selectin-mediated adhesion. These results indicate that L-selectin prefers carbohydrate 6-sulfation much more than tyrosine sulfation, whereas P-selectin favors tyrosine **sulfation** in the PSGL-1 molecule far more than carbohydrate 6- ***sulfation*** . ***E*** - **selectin**-mediated adhesion was **sulfation-independent** requiring only Fuc-T VII, and thus the three members of the selectin family have distinct requirements for ligand sulfation.

7/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012896636 BIOSIS NO.: 200100068475
A distinct glycoform of CD44 is an L-selectin ligand on human hematopoietic cells
AUTHOR: Dimitroff Charles J; Lee Jack Y; Fuhlbrigge Robert C; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Avenue Louis Pasteur,

Room 671, Boston, MA, 02115, USA**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (25): p13841-13846 December 5, 2000 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We previously have obtained operational evidence of a hematopoietic cell L-selectin ligand expressed on normal human hematopoietic cells and on leukemic blasts. Using a technique developed in our laboratory for analyzing and identifying adhesion molecules, we show here that hematopoietic cell L-selectin ligand is a specialized glycoform of CD44. This ***L*** - ***selectin*** ligand activity of CD44 requires sialofucosylated N-linked glycans and is **sulfation-** ***independent*** . These data provide important insights on the structural biology of CD44 and reveal a role for this protein as an L-selectin ligand on human hematopoietic cells.

7/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012473562 BIOSIS NO.: 200000191875
Modification of P-selectin glycoprotein ligand-1 with a natural killer cell-restricted sulfated lactosamine creates an alternate ligand for L-selectin
AUTHOR: Andre Pascale; Spertini Olivier; Guia Sophie; Rihet Pascal; Dignat-George Francoise; Brailly Herve; Sampol Jose; Anderson Paul J; Vivier Eric (Reprint)
AUTHOR ADDRESS: Centre d'Immunologie de Marseille-Luminy, Institut National de la Sante et de la Recherche Medicale/Centre National de la Recherche Scientifique, 13288, Marseille Cedex 09, France**France
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (7): p3400-3405 March 28, 2000 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Natural killer (NK) cells are components of the innate immune system that can recognize and kill virally infected cells, tumor cells, and allogeneic cells without prior sensitization. NK cells also elaborate cytokines (e.g., interferon-gamma and tumor necrosis factor-alpha) and chemokines (e.g., macrophage inflammatory protein-1alpha) that promote the acquisition of antigen-specific immunity. NK cell differentiation is accompanied by the cell surface expression of a mucin-like glycoprotein bearing an NK cell-restricted keratan sulfate-related lactosamine carbohydrate, the PEN5 epitope. Here, we report that PEN5 is a post-translational modification of P-selectin glycoprotein ligand-1 (PSGL-1). The PEN5 epitope creates on PSGL-1 a unique binding site for **L-selectin**, which is **independent** of PSGL-1 tyrosine ***sulfation*** . On the surface of NK cells, the expression of PEN5 is coordinated with the disappearance of L-selectin and the up-regulation of Killer cell Ig-like Receptors (KIR). These results indicate that NK cell differentiation is accompanied by the acquisition of a unique carbohydrate, PEN5, that can serve as part of a combination code to deliver KIR+ NK cells to specific tissues.

7/7/4 (Item 4 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

0010878764 BIOSIS NO.: 199799512824
A hematopoietic cell L-selectin ligand exhibits sulfate-independent binding activity
AUTHOR: Sackstein Robert (Reprint); Fu Ling; Allen Katrina L
AUTHOR ADDRESS: Div. Bone Marrow Transplantation, H. Lee Moffitt Cancer Center Research Inst., 12902 Magnolia Dr., Tampa, FL 33612, USA**USA
JOURNAL: Blood 89 (8): p2773-2781 1997 1997
ISSN: 0006-4971
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: L-selectin is a leukocyte cell-surface glycoprotein that mediates adhesive interactions between circulating cells and vascular endothelium. All endothelial ligands of L-selectin characterized to date are glycoproteins that require sulfation for activity and share reactivity with MECA 79, a monoclonal antibody that recognizes a sulfate-dependent epitope involved in L-selectin attachment. We have recently identified by functional assay a glycoprotein L-selectin ligand expressed on the human hematopoietic cell line KG1a. We report here that this ligand is not recognized by MECA 79 and that it retains binding activity after metabolic inhibition of ***sulfation*** by chlorate. A native membrane **L-selectin** ligand exhibiting sulfate-**independent** function has not been described previously. Identification of this novel ligand on a nonendothelial cell type suggests that structural determinants conferring L-selectin binding may vary in a cell- and tissue-specific manner.

7/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009577924 BIOSIS NO.: 199598045757
Sulfation-dependent recognition of high endothelial venules (HEV)-ligands by L-selection and MECA 79, an adhesion-blocking monoclonal antibody
AUTHOR: Hemmerich Stefan; Butcher Eugene C; Rosen Steven D (Reprint)
AUTHOR ADDRESS: Dep. Anat., Univ. Calif., San Francisco, CA 94143-0452, USA **USA
JOURNAL: Journal of Experimental Medicine 180 (6): p2219-2226 1994 1994
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: L-selectin is a lectin-like receptor that mediates the attachment of lymphocytes to high endothelial venules (HEV) of lymph nodes during the process of lymphocyte recirculation. Two sulfated, mucin-like glycoproteins known as Sgp50/GlyCAM-1 and Sgp90/CD34 have previously been identified as HEV-associated ligands for L-selectin. These proteins were originally detected with an L-selectin/Ig chimera called LEC-IgG. GlyCAM-1 and CD34 are also recognized by an antiperipheral node addressin (PNAd) mAb called MECA 79, which blocks L-selectin-dependent adhesion and selectively stains lymph node HEV. The present study compares the requirements for the binding of MECA 79 and LEC-IgG to HEV-ligands. Whereas desialylation of GlyCAM-1 and CD34 drastically reduced binding to LEC-IgG, this treatment enhanced the binding of GlyCAM-1 to MECA 79. In contrast, the binding of both MECA 79 and LEC-IgG to GlyCAM-1 and CD34 was greatly decreased when the sulfation of these ligands was reduced with chlorate, a metabolic inhibitor of sulfation. Because MECA 79 stains HEV-like vessels at various sites of inflammation, recognition by

L-selectin of ligands outside of secondary lymphoid organs may depend on ***sulfation*** . In addition to their reactivity with GlyCAM-1 and CD34, both MECA 79 and LEC-IgG recognize an **independent** molecule of apprx 200 kD in a sulfate-dependent manner. Thus, this molecule, which we designate Sgp200, is an additional ligand for L-selectin.

7/7/6 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

05941946 EMBASE No: 1994357259
Sulfation-dependent recognition of high endothelial venules (HEV)-ligands by L-selectin and MECA 79, an adhesion-blocking monoclonal antibody
Hemmerich S.; Butcher E.C.; Rosen S.D.
Department of Anatomy, University of California,San Francisco, CA 94143-0452 United States
Journal of Experimental Medicine (J. EXP. MED.) (United States) 1994, 180/6 (2219-2226)
CODEN: JEMEA ISSN: 0022-1007
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

L-selectin is a lectin-like receptor that mediates the attachment of lymphocytes to high endothelial venules (HEV) of lymph nodes during the process of lymphocyte recirculation. Two sulfated, mucin-like glycoproteins known as Sgp50/GlyCAM-1 and Sgp90/CD34 have previously been identified as HEV-associated ligands for L-selectin. These proteins were originally detected with an L-selectin/Ig chimera called LEC-IgG. GlyCAM-1 and CD34 are also recognized by an anti-peripheral node addressin (PNAd) mAb called MECA 79, which blocks L-selectin-dependent adhesion and selectively stains lymph node HEV. The present study compares the requirements for the binding of MECA 79 and LEC-IgG to HEV-ligands. Whereas desialylation of GlyCAM-1 and CD34 drastically reduced binding to LEC-IgG, this treatment enhanced the binding of GlyCAM-1 to MECA 79. In contrast, the binding of both MECA 79 and LEC-IgG to GlyCAM-1 and CD34 was greatly decreased when the sulfation of these ligands was reduced with chlorate, a metabolic inhibitor of sulfation. Because MECA 79 stains HEV-like vessels at various sites of inflammation, recognition by L-selectin of ligands outside of secondary lymphoid organs may depend on ***sulfation*** . In addition to their reactivity with GlyCAM-1 and CD34, both MECA 79 and LEC-IgG recognize an ***independent*** molecule of ~200 kD in a sulfate-dependent manner. Thus, this molecule, which we designate Sgp200, is an additional ligand for L-selectin.

7/7/7 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11990930 PMID: 12068018
Distinct sulfation requirements of selectins disclosed using cells that support rolling mediated by all three selectins under shear flow. L-selectin prefers carbohydrate 6-sulfation totyrosine sulfation, whereas p-selectin does not.
Kanamori Akiko; Kojima Naoya; Uchimura Kenji; Muramatsu Takashi; Tamatani Takuya; Berndt Michael C; Kansas Geoffrey S; Kannagi Reiji
Program of Molecular Pathology, Aichi Cancer Center, Research Institute, Nagoya 464-8681, Japan.
Journal of biological chemistry (United States) Sep 6 2002, 277 (36) p32578-86, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant Number: HL55647; HL; NHLBI
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed
l- and P-selectin are known to require sulfation in their ligand molecules. We investigated the significance of carbohydrate 6-sulfation and tyrosine sulfation in selectin-mediated cell adhesion. COS-7 cells were genetically engineered to express P-selectin glycoprotein ligand-1 (PSGL-1) or its mutant in various combinations with 6-O-sulfotransferase (6-Sul-T) and/or alpha1-->3fucosyltransferase VII (Fuc-T VII). The cells transfected with PSGL-1, 6-Sul-T, and Fuc-T VII cDNAs supported rolling mediated by all three selectins and provided the best experimental system so far to estimate kinetic parameters in selectin-mediated cell adhesion for all three selectins using the identical rolling substrate and to compare the ligand specificity of each selectin. L-selectin-mediated rolling was drastically impaired if the cells lacked carbohydrate 6-sulfation elaborated by 6-Sul-T, but not affected when PSGL-1 was replaced with a mutant lacking three tyrosine residues at its NH(2) terminus. L-selectin-mediated adhesion was also hardly affected by mocarhagin treatment of the cells, which cleaved a short peptide containing sulfated tyrosine residues from PSGL-1. In contrast, P-selectin-mediated rolling was abolished when PSGL-1 was either mutated or cleaved by mocarhagin at its NH(2) terminus, whereas the cells expressing PSGL-1 and Fuc-T VII but not 6-Sul-T showed only a modest decrease in P-selectin-mediated adhesion. These results indicate that L-selectin prefers carbohydrate 6-sulfation much more than tyrosine sulfation, whereas P-selectin favors tyrosine **sulfation** in the PSGL-1 molecule far more than carbohydrate 6-
sulfation . ***E*** - ***selectin*** -mediated adhesion was
sulfation-independent requiring only Fuc-T VII, and thus the three members of the selectin family have distinct requirements for ligand sulfation.
Record Date Created: 20020902
Record Date Completed: 20021029
Date of Electronic Publication: 20020614
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S6	7	S5(20N)(INDEPENDENT)
S7	7	RD S6 (unique items)

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 9/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0013806841   BIOSIS NO.: 200200400352
Expression of selectin ligands on blasts from human acute leukemias
AUTHOR: Sackstein R; Dimitroff C; Lee J
JOURNAL: Experimental Hematology (Charlottesville)  30 (6 Supplement 1): p
81 June, 2002 2002
MEDIUM: print
CONFERENCE/MEETING: 31st Annual Meeting of the International Society for
Experimental Hematology  Montreal, Quebec, Canada  July 05-09, 2002;
20020705
ISSN: 0301-472X
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DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

9/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013627075 BIOSIS NO.: 200200220586
Homing and hematopoiesis: **HCELL** is the principal E-selectin and L-selectin ligand of human hematopoietic stem cells
AUTHOR: Sackstein Robert (Reprint); Dimitroff Charles J (Reprint); Lee Jack Y (Reprint); Fuhlbrigge Robert C (Reprint); Parmar Kalindi; Mauch Peter M ; Sandmaier Brenda M
AUTHOR ADDRESS: Dermatology and Medicine, Brigham and Women's Hospital, Boston, MA, USA**USA
JOURNAL: Blood 98 (11 Part 1): p710a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The selectins are becoming increasingly recognized for playing key roles in hematopoiesis. The endothelial selectins, E- and P-selectin, are both constitutively expressed on bone marrow (BM) microvascular endothelium, where they help mediate hematopoietic progenitor cell (HPC) migration into BM. Expression of the leukocyte selectin, L-selectin, on human CD34+ HPCs is associated with higher clonogenic activity in in vitro assays and faster engraftment following BM transplantation. Human HPCs also express PSGL-1, a ligand for all three selectins, however, paradoxically, engagement of PSGL-1 appears to inhibit clonogenic activity of human HPCs. These published data, collectively, have prompted us to explore the structure and distribution of selectin ligands expressed on human HPCs. Utilizing a new shear-based adhesion assay system developed in our laboratory, we have analyzed the cell surface glycoproteins of normal human HPCs that mediate L-, E- and P-selectin binding. Normal BM cells were separated into various lineage- and lineage+ subsets by magnetic bead sorting, and also sorted by flow cytometry of "side-population" cells following Hoechst dye staining. Cell membrane proteins were resolved into component bands by SDS-PAGE, then blotted onto PVDF. The blot was then placed in a flow chamber apparatus, and L-selectin+lymphocytes or stably transfected CHO cells bearing E- or P-selectin (designated CHO-E and CHO-P, respectively) were introduced into the chamber under controlled flow conditions. Adhesive interactions between cells in flow and immobilized (blot) proteins were visualized by video microscopy. CHO-P adhesive interactions occurred only at bands corresponding to PSGL-1. Adhesive interactions using lymphocytes and CHO-E were also observed at bands corresponding to PSGL-1, but significantly more L- and E-selectin ligand activity was observed at a band of apprx100,000 mw, operationally named "Hematopoietic Cell E-/L-selectin Ligand" (***HCELL***). Mass spectroscopy analysis of this protein, confirmed by immunopurification, revealed that this E- and L-selectin ligand is a previously unrecognized glycoform of a well-characterized glycoprotein, CD44. In shear-based adhesion assays of purified protein or of protein expressed naturally on cell membranes, **HCELL** displays >5-fold more avidity for E- and for L-selectin compared to PSGL-1. Though CD44 is broadly expressed among normal human BM marrow cells, **HCELL** is expressed only on lineage- cells: its expression is characteristic of CD34+ cells, with highest expression in

CD38-/lin- cells. Additionally, ***HCELL*** is expressed on CD34+ and CD34- subsets of "side-population" cells. The distinctive, restricted expression of **HCELL** among the subsets comprising the human hematopoietic "stem" cell and its marked avidity for E- and L-selectin supports a role for this unique glycoform of CD44 as a BM "homing receptor" as well as being the principal ligand mediating L-selectin-dependent cell-cell adhesive events within the BM microenvironment.

9/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013567681 BIOSIS NO.: 200200161192
Differential L-selectin binding activities of human hematopoietic cell L-selectin ligands, **HCELL** and PSGL-1
AUTHOR: Dimitroff Charles J; Lee Jack Y; Schor Kenneth S; Sandmaier Brenda M; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, Harvard Skin Disease Research Center, 77 Ave. Louis Pasteur, Boston, MA, 02115, USA**USA
JOURNAL: Journal of Biological Chemistry 276 (50): p47623-47631 December 14, 2001 2001
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Expression of L-selectin on human hematopoietic cells (HC) is associated with a higher proliferative activity and a more rapid engraftment after hematopoietic stem cell transplantation. Two L-selectin ligands are expressed on human HCs, P-selectin glycoprotein ligand-1 (PSGL-1) and a specialized glycoform of CD44 (hematopoietic cell E- and L-selectin ligand, ***HCELL***). Although the structural biochemistry of **HCELL** and PSGL-1 is well characterized, the relative capacity of these molecules to mediate L-selectin-dependent adhesion has not been explored. In this study, we examined under shear stress conditions L-selectin-dependent leukocyte adhesive interactions mediated by **HCELL** and PSGL-1, both as naturally expressed on human HC membranes and as purified molecules. By utilizing both Stamper-Woodruff and parallel-plate flow chamber assays, we found that **HCELL** displayed a 5-fold greater capacity to support L-selectin-dependent leukocyte adherence across a broad range of shear stresses compared with that of PSGL-1. Moreover, L-selectin-mediated leukocyte binding to immunopurified **HCELL** was consistently >5-fold higher than leukocyte binding to equivalent amounts of PSGL-1. Taken together, these data indicate that **HCELL** is a more avid L-selectin ligand than PSGL-1 and may be the preferential mediator of L-selectin-dependent adhesive interactions among human HCs in the bone marrow.

9/7/4 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

137017447 CA: 137(2)17447w PATENT
Hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
INVENTOR(AUTHOR): Sackstein, Robert
LOCATION: USA
ASSIGNEE: The Brigham and Women's Hospital, Inc.
PATENT: PCT International ; WO 200244342 A2 DATE: 20020606
APPLICATION: WO 2001US51014 (20011018) *US PV240987 (20001018) *US

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  SECTION:
CA209016 Biochemical Methods
CA201XXX Pharmacology
CA203XXX Biochemical Genetics
CA206XXX General Biochemistry
CA213XXX Mammalian Biochemistry
CA214XXX Mammalian Pathological Biochemistry
  IDENTIFIERS: hematopoietic cell selectin ligand peptide sequence cancer
drug immunoassay
  DESCRIPTORS:
Ricins...
    A; hematopoietic cell E-selection/L-selectin ligand polypeptides and
    methods of use thereof
Blood...
    cancer; hematopoietic cell E-selection/L-selectin ligand polypeptides
    and methods of use thereof
Muscular dystrophy...
    congenital; hematopoietic cell E-selection/L-selectin ligand
    polypeptides and methods of use thereof
Bond...
    covalent; hematopoietic cell E-selection/L-selectin ligand polypeptides
    and methods of use thereof
Toxins...
    diphtheria; hematopoietic cell E-selection/L-selectin ligand
    polypeptides and methods of use thereof
Hematopoiesis...
    disorders; hematopoietic cell E-selection/L-selectin ligand
    polypeptides and methods of use thereof
Selectins...
    E-; hematopoietic cell E-selection/L-selectin ligand polypeptides and
    methods of use thereof
Transplant and Transplantation...
    engraftment potential; hematopoietic cell E-selection/L-selectin ligand
    polypeptides and methods of use thereof
Pseudomonas...
    exotoxin; hematopoietic cell E-selection/L-selectin ligand polypeptides
    and methods of use thereof
Disease,animal...
    genetic; hematopoietic cell E-selection/L-selectin ligand polypeptides
    and methods of use thereof
Sialic acids...
    groups; hematopoietic cell E-selection/L-selectin ligand polypeptides
    and methods of use thereof
Antibodies...
    HCELL; hematopoietic cell E-selection/L-selectin ligand polypeptides
    and methods of use thereof
Hematopoietic precursor cell... Ligands... Inflammation... Neoplasm...
Mammalia... Protein sequences... Immunoassay... CD44(antigen)... Antibodies
... Immobilization,molecular... Shear stress... Blood analysis...
Erythrocyte... Bone marrow... Affinity... Nucleic acids... Human... Drug
screening... Parkinson's disease... Diabetes mellitus... Liver,disease...
Toxins... Genetic methods... Molecular recognition... Molecular association
...
    hematopoietic cell E-selection/L-selectin ligand polypeptides and
    methods of use thereof
Heart,disease...
    infarction; hematopoietic cell E-selection/L-selectin ligand
    polypeptides and methods of use thereof
Selectins...
    L-; hematopoietic cell E-selection/L-selectin ligand polypeptides and

methods of use thereof
Antibodies...
monoclonal, GECA-452; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
Carbohydrates,processes...
N-linked groups; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
Proteins...
PAP (pokeweed antiviral protein); hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
Peptides,uses...
polypeptides, glycosylated; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
Therapy...
stem cell; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
Hematopoietic precursor cell...
stem; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
Brain,disease...
stroke; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
CAS REGISTRY NUMBERS:
434529-63-2 amino acid sequence; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
83534-39-8 9001-67-6 111070-05-4 9033-07-2 9032-92-2 95787-44-3 75037-46-6 hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
2438-80-4 moieties; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
434530-60-6 434530-61-7 434530-62-8 434530-63-9 434530-64-0 434530-65-1 unclaimed sequence; hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof
? s cd44
S10 15702 CD44
? s s10(20n)(sulfation)
15702 S10
15453 SULFATION
S11 40 S10(20N)(SULFATION)
? rd s11
...completed examining records
S12 15 RD S11 (unique items)
? t s12/7/all

12/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013995233 BIOSIS NO.: 200200588744
TNF-alpha increases the carbohydrate **sulfation** of **CD44**:
Induction of 6-sulfo N-acetyl lactosamine on N- and O-linked glycans
AUTHOR: Delcommenne Marc; Kannagi Reiji; Johnson Pauline (Reprint)
AUTHOR ADDRESS: Section of Bone Marrow Transplantation, Rush Presbyterian-St. Luke's Medical Center, Chicago, IL, 60612, USA**USA
JOURNAL: Glycobiology 12 (10): p613-622 October, 2002 2002
MEDIUM: print
ISSN: 0959-6658
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **CD44** and **sulfation** have both been implicated in leukocyte adhesion. In monocytes, the inflammatory cytokine tumor necrosis factor alpha (TNF-alpha) stimulates **CD44 sulfation**,

and this correlates with the induction of **CD44**-mediated adhesion events. However, little is known about the ***sulfation*** of ***CD44*** or its induction by inflammatory cytokines. We determined that TNF-alpha induces the carbohydrate ***sulfation*** of ***CD44*** . ***CD44*** was established as a major sulfated cell surface protein on myeloid cells. In the SR91 myeloid cell line, the majority of **CD44 sulfation** was attributed to the glycosaminoglycan chondroitin sulfate. However, TNF-alpha stimulation increased **CD44 sulfation** two- to threefold, largely attributed to the increased **sulfation** of N- and O-linked glycans on ***CD44*** . Therefore, TNF-alpha induced a decrease in the percentage of **CD44 sulfation** due to chondroitin sulfate and an increase due to N- and O-linked ***sulfation*** . Furthermore, TNF-alpha induced the expression of 6-sulfo N-acetyl lactosamine (LacNAc)/Lewis x on these cells, which was detected by a monoclonal antibody after neuraminidase treatment. This 6-sulfo LacNAc/Lewis x epitope was induced on N-linked and (to a lesser extent) on O-linked glycans present on CD44. This demonstrates that ***CD44*** is modified by sulfated carbohydrates in myeloid cells and that TNF-alpha modifies both the type and amount of carbohydrate **sulfation** occurring on ***CD44*** . In addition, it demonstrates that TNF-alpha can induce the expression of 6-sulfo N-acetyl glucosamine on both N- and O-linked glycans of CD44 in myeloid cells.

12/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013693601 BIOSIS NO.: 200200287112
Oversulfated chondroitin/dermatan sulfates containing GlcAbeta1/IdoAalpha1-3GalNAc(4,6-O-disulfate) interact with L- and P-selectin and chemokines
AUTHOR: Kawashima Hiroto (Reprint); Atarashi Kazuyuki; Hirose Mayumi; Hirose Jun; Yamada Shuhei; Sugahara Kazuyuki; Miyasaka Masayuki
AUTHOR ADDRESS: Glycobiology Program, Cancer Research Center, Burnham Institute, La Jolla, CA, 92037, USA**USA
JOURNAL: Journal of Biological Chemistry 277 (15): p12921-12930 April 12, 2002 2002
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We previously reported that versican, a large chondroitin/dermatan sulfate (CS/DS) proteoglycan, interacts through its CS/DS chains with adhesion molecules L- and P-selectin and **CD44**, as well as chemokines. Here, we have characterized these interactions further. Using a metabolic inhibitor of ***sulfation*** , sodium chlorate, we show that the interactions of the CS/DS chains of versican with L- and P-selectin and chemokines are **sulfation**-dependent but the interaction with ***CD44*** is ***sulfation*** -independent. Consistently, versican's binding to L- and P-selectin and chemokines is specifically inhibited by oversulfated CS/DS chains containing GlcAbeta1-3GalNAc(4,6-O-disulfate) or IdoAalpha1-3GalNAc(4,6-O-disulfate), but its binding to CD44 is inhibited by all the CS/DS chains, including low-sulfated and unsulfated ones. Affinity and kinetic analyses using surface plasmon resonance revealed that the oversulfated CS/DS chains containing GlcAbeta1/IdoAalpha1-3GalNAc(4,6-O-disulfate) bind directly to selectins and chemokines with high affinity (Kd 21.1 to 293 nM). In addition, a tetrasaccharide fragment of repeating GlcAbeta1-3GalNAc(4,6-O-disulfate) units directly interacts with L- and P-selectin and chemokines and oversulfated CS/DS chains containing

GlcAbeta1/IdoAalpha1-3GalNAc(4,6-O-disulfate) inhibit chemokine-induced Ca2+ mobilization. Taken together, our results show that oversulfated CS/DS chains containing GlcAbeta1/IdoAalpha1-3GalNAc(4,6-O-disulfate) are recognized by L- and P-selectin and chemokines, and imply that these chains are important in selectin- and/or chemokine-mediated cellular responses.

12/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012896636 BIOSIS NO.: 200100068475
A distinct glycoform of CD44 is an L-selectin ligand on human hematopoietic cells
AUTHOR: Dimitroff Charles J; Lee Jack Y; Fuhlbrigge Robert C; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Avenue Louis Pasteur, Room 671, Boston, MA, 02115, USA**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (25): p13841-13846 December 5, 2000 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We previously have obtained operational evidence of a hematopoietic cell L-selectin ligand expressed on normal human hematopoietic cells and on leukemic blasts. Using a technique developed in our laboratory for analyzing and identifying adhesion molecules, we show here that hematopoietic cell L-selectin ligand is a specialized glycoform of ***CD44*** . This L-selectin ligand activity of ***CD44*** requires sialofucosylated N-linked glycans and is **sulfation** -independent. These data provide important insights on the structural biology of **CD44** and reveal a role for this protein as an L-selectin ligand on human hematopoietic cells.

12/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012865450 BIOSIS NO.: 200100037289
Characterization of TNF-alpha induced **sulfation** of **CD44**
AUTHOR: Delcommenne M (Reprint); Maiti A (Reprint); Johnson P (Reprint)
AUTHOR ADDRESS: Department of Microbiology and Immunology, University of British Columbia, No. 300-6174 University Boulevard, Vancouver, BC, V6T 1Z3, Canada**Canada
JOURNAL: FASEB Journal 14 (6): pA1142 April 20, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: Joint Annual Meeting of the American Association of Immunologists and the Clinical Immunology Society Seattle, Washington, USA May 12-16, 2000; 20000512
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

12/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012625526 BIOSIS NO.: 200000343839
Oncostatin M and transforming growth factor-beta1 induce post-translational modification and hyaluronan binding to CD44 in lung-derived epithelial tumor cells
AUTHOR: Cichy Joanna (Reprint); Pure Ellen (Reprint)
AUTHOR ADDRESS: Wistar Institute, Philadelphia, PA, 19104, USA**USA
JOURNAL: Journal of Biological Chemistry 275 (24): p18061-18069 June 16, 2000 2000
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD44, a receptor for hyaluronan (HA), has been implicated in tumor growth and metastasis. Most CD44-positive cells fail to exhibit constitutive HA receptor function but CD44-mediated HA binding on hematopoetic cells can be induced by antibody cross-linking of the receptor and by physiologic stimuli, including cytokines. We now demonstrate that oncostatin M (OSM) and transforming growth factor-beta1, cytokines known to regulate the growth of tumor cells, stimulate HA binding in lung epithelial-derived tumor cells. In lung epithelial-derived tumor cells, cytokine-induced binding resulted from post-translational modification of the receptor. OSM-induced HA binding was associated with a reduction in N-linked carbohydrate content of ***CD44*** . In addition, OSM induced HA binding via a novel mechanism requiring **sulfation** of chondroitin sulfate chains linked to ***CD44*** . The mechanism underlying transforming growth factor-beta1 induced HA binding was distinct from the effects of OSM. The data presented indicate that modulation of the glycosylation and **sulfation** of **CD44** by cytokines provides mechanisms for regulating cell adhesion during tumor growth and metastasis.

12/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012396099 BIOSIS NO.: 200000114412
A role for the cell adhesion molecule **CD44** and **sulfation** in leukocyte-endothelial cell adhesion during an inflammatory response?
AUTHOR: Johnson Pauline (Reprint); Maiti Arpita; Brown Kelly L; Li Ruihong
AUTHOR ADDRESS: Department of Microbiology and Immunology, University of British Columbia, No. 300 - 6174 University Boulevard, Vancouver, B.C., V6T 1Z3, Canada**Canada
JOURNAL: Biochemical Pharmacology 59 (5): p455-465 March 1, 2000 2000
MEDIUM: print
ISSN: 0006-2952
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD44 is a widely expressed cell adhesion molecule that has been implicated in a variety of biological processes including lymphopoiesis, angiogenesis, wound healing, leukocyte extravasation at inflammatory sites, and tumor metastasis. The adhesive function of CD44, like other molecules involved in inducible adhesion, is tightly regulated. Post-translational modifications, isoform expression, aggregation state, and protein associations all can affect the ligand binding properties of CD44, and these can vary depending on the cell type and the activation state of the cell. The most extensively characterized ligand for CD44 is hyaluronan, a component of the extracellular matrix. Interactions between CD44 and hyaluronan can mediate both cell-cell and cell-extracellular matrix adhesion. In the immune system, both the selectin molecules and

CD44 have been implicated in the initial binding of leukocytes to endothelial cells at an inflammatory site. ***Sulfation*** is required for selectin-mediated leukocyte-endothelial cell interactions, and, recently, inducible **sulfation** also was shown to regulate **CD44** -mediated leukocyte adhesion to endothelial cells. ***Sulfation*** , therefore, may be important in the regulation of cell adhesion at inflammatory sites. In this commentary we have reviewed the molecular aspects of CD44 and the mechanisms that regulate its binding to hyaluronan. In addition, we have summarized the role of CD44 and hyaluronan in mediating leukocyte-endothelial cell interactions and have discussed how this interaction may be regulated. Finally, we examined the potential role of **sulfation** as an inducible means to regulate **CD44**-mediated leukocyte adhesion and as a more general mechanism to regulate leukocyte-endothelial cell interactions.

12/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012015581 BIOSIS NO.: 199900275241
TNFalpha and IL-4 regulation of hyaluronan binding to monocyte CD44 involves posttranslational modification of CD44
AUTHOR: Levesque Marc C (Reprint); Haynes Barton F
AUTHOR ADDRESS: Duke University Medical Center, Durham, NC, 27710, USA**USA
JOURNAL: Cellular Immunology 193 (2): p209-218 May 1, 1999 1999
MEDIUM: print
ISSN: 0008-8749
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Our previous studies have identified TNFalpha as a positive regulator and IL-4 as a negative regulator of human monocyte CD44-HA binding. In order to determine the mechanisms of IL-4- and TNFalpha-mediated regulation of monocyte HA binding, we measured HA binding and CD44 expression on peripheral blood monocytes following monocyte treatment with TNFalpha or IL-4, as well as following monocyte treatment with inhibitors of protein synthesis, N- and O-linked glycosylation, and chondroitin ***sulfation*** . IL-4 decreased ***CD44*** -HA binding on monocytes initially treated with TNFalpha. Similarly, pretreatment of monocytes with IL-4 prevented subsequent TNFalpha-mediated HA binding. Cycloheximide (protein synthesis inhibitor), tunica-mycin (N-linked glycosylation inhibitor), and beta-D-xylo-side (chondroitin **sulfation** inhibitor) all inhibited IL-4-mediated downregulation of TNFalpha-induced monocyte HA binding. Western blot analysis of **CD44** from TNFalpha-treated monocytes revealed a 5-10 Mr decrease in the standard isoform of CD44. In contrast, IL-4 treatment of monocytes inhibited CD44-HA binding and reversed the 5- to 10-kDa decrease in monocyte CD44 Mr. Finally, studies with F10.44.2, a CD44 mab that enhances CD44-HA binding, indicated that IL-4 treatment of monocytes not only diminished constitutive HA binding, but also diminished CD44 mab-induced HA binding. Taken together, these data suggested that IL-4-mediated inhibition of TNFalpha-induced monocyte HA binding was dependent not only on protein synthesis, but also on N-linked glycosylation and chondroitin-sulfate modification of either CD44 or, alternatively, another monocyte protein(s) that may regulate the ability of CD44 to bind HA.

12/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011987228 BIOSIS NO.: 199900246888
Heparan sulfate composition of alternatively spliced CD44 fusion proteins
AUTHOR: Piepkorn Michael; Hovingh Peter; Bennett Kelly L; Linker Alfred (Reprint)
AUTHOR ADDRESS: University of Washington, Seattle, WA, USA**USA
JOURNAL: Biochemical and Biophysical Research Communications 257 (3): p 839-842 April 21, 1999 1999
MEDIUM: print
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Prior analyses of recombinant CD44 fusion proteins have indicated that combinatorial splicing of variant exons exerts distal effects on chondroitin sulfate content and structure, which may regulate the biological properties of the respective CD44 isoforms. The consequences of splicing of variant exons V4-7 on the heparan sulfate moieties were therefore examined, utilizing recombinant chimeras containing exons V3 and V8-10, engineered with or without exons V4-7 and expressed as Ig fusion proteins in COS cells. Splicing of exons V4-7, though they contain no consensus motifs for glycosaminoglycan assembly, resulted in markedly increased polymer ***sulfation*** levels of the heparan sulfates. The sulfate groups of both the **CD44** V3-10 and V3,8-10 isoforms occurred as di- and tri-sulfated dissacharide units and were restricted to one N-sulfated block domain within the polymers. Compared to native human keratinocyte CD44, the recombinant heparan sulfates were relatively low in sulfate content. Our data indicate that variant exon V4-7 splicing exerts distal effects on the composition of this glycosaminoglycan. These effects may regulate those functions that are mediated through the heparan sulfate moieties, such as the binding of growth factors.

12/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011756276 BIOSIS NO.: 199900015936
Regulation of **CD44** mediated adhesion by **sulfation**
AUTHOR: Maiti A (Reprint); Maki G; Johnson P (Reprint)
AUTHOR ADDRESS: Dep. Microbiol. and Immunol., Univ. British Columbia, Vancouver, BC, Canada**Canada
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p198A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

12/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011742334 BIOSIS NO.: 199900001994
TNF-alpha induction of **CD44**-mediated leukocyte adhesion by **sulfation**
AUTHOR: Maiti Arpita; Maki Giutta; Johnson Pauline (Reprint)
AUTHOR ADDRESS: Dep. Microbiol. Immunol., Univ. B.C., 300-6174 University Blvd., Vancouver, BC V6T 1Z3, Canada**Canada
JOURNAL: Science (Washington D C) 283 (5390): p941-943 Oct. 30, 1998 1998

MEDIUM: print
ISSN: 0036-8075
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Regulation of cell adhesion is important for immune system function. CD44 is a tightly regulated cell adhesion molecule present on leukocytes and implicated in their attachment to endothelium during an inflammatory immune response. The proinflammatory cytokine tumor necrosis factor-alpha, but not interferon-gamma, was found to convert **CD44** from its inactive, nonbinding form to its active form by inducing the ***sulfation*** of ***CD44*** . This posttranslational modification was required for **CD44**-mediated binding to the extracellular matrix component hyaluronan and to vascular endothelial cells. ***Sulfation*** is thus a potential means of regulating **CD44**-mediated leukocyte adhesion at inflammatory sites.

12/7/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011474148 BIOSIS NO.: 199800268395
Analysis of CD44 interactions with hyaluronan in murine L cell fibroblasts deficient in glycosaminoglycan synthesis: A role for chondroitin sulfate
AUTHOR: Esford Lesley E; Maiti Arpita; Bader Sharon A; Tufaro Frank; Johnson Pauline (Reprint)
AUTHOR ADDRESS: Dep. Microbiol. Immunol., Univ. B.C., Vancouver, BC V6T 1Z3, Canada**Canada
JOURNAL: Journal of Cell Science 111 (7): p1021-1029 April, 1998 1998
MEDIUM: print
ISSN: 0021-9533
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: CD44 is a widely expressed cell adhesion molecule that binds the extracellular matrix component, hyaluronan, in a tightly regulated manner. Previous studies have shown that the CD44-hyaluronan interaction is affected by changes in the glycosylation state of CD44. In this study, we take advantage of several well-characterized murine L cell mutants defective in heparan sulfate synthesis (gro2C cells), heparan sulfate and chondroitin sulfate synthesis (sog9 cells), and glycosaminoglycan and oligosaccharide processing (sog8 cells) to assess the effects of these defects on the hyaluronan binding ability of CD44. In parental L cells and gro2C cells, CD44 was induced to bind hyaluronan after addition of the activating, anti-CD44 monoclonal antibody, IRAWB 14. By contrast, no inducible binding was observed in sog9 cells. Treatment of L cells with sodium chlorate, an inhibitor of sulfation, also abolished inducible hyaluronan binding. However, inducible and some constitutive hyaluronan binding was observed in sog8 cells. This indicates that ***sulfation*** and, in particular, the addition of chondroitin sulfate are required for inducible hyaluronan binding by ***CD44*** in L cells. However, in the absence of fully processed oligosaccharides, chondroitin sulfate is not essential for hyaluronan binding, indicating that the effect of chondroitin sulfate is dependent upon the glycosylation state of the cell. Thus, in addition to glycosylation, chondroitin sulfate biosynthesis is an important post-translational modification that can affect the hyaluronan binding ability of CD44.

12/7/12 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

11367187 EMBASE No: 2001381457
Role of **sulfation** in **CD44**-mediated hyaluronan binding induced by inflammatory mediators in human CD14SUP+ peripheral blood monocytes
Brown K.L.; Maiti A.; Johnson P.
Dr. P. Johnson, Department of Microbiology, University of British Columbia, Number 300-6174 University Boulevard, Vancouver, BC V6T IZ3 Canada
AUTHOR EMAIL: pauline@interchange.ubc.ca
Journal of Immunology (J. IMMUNOL.) (United States) 01 NOV 2001, 167/9 (5367-5374)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 44

Activation of T cells by Ag or stimulation of monocytes with inflammatory cytokines induces CD44 to bind to hyaluronan (HA), an adhesion event implicated in leukocyte-leukocyte, leukocyte-endothelial cell, and leukocyte-stromal cell interactions. We have previously shown that TNF-alpha induces **CD44 sulfation** in a leukemic cell line, which correlated with the induction of HA binding and **CD44**-mediated adhesion. In this study, we establish that TNF-alpha and IFN-gamma induce HA binding and the **sulfation** of **CD44** in CD14SUP+ PBMC, whereas no induced HA binding or **CD44 sulfation** was observed in CD14SUP- PBMC stimulated with TNF-alpha. Treatment of cells with NaCIOSUB3, an inhibitor of **sulfation**, prevented HA binding in a significant percentage of CD14SUP+ PBMC induced by TNF-alpha, LPS, IL-1beta, or IFN-gamma. Furthermore, stimulation with TNF-alpha or IFN-gamma in the presence of NaCIOSUB3 reduced the ability of isolated CD44H to bind HA, demonstrating a direct effect of CD44H sulfation on HA binding. In contrast, the transient induction of HA binding in T cells by PHA was not affected by NaCIOSUB3, suggesting that activated T cells do not use sulfation as a mechanism to regulate HA binding. Overall, these results demonstrate that inducible sulfation of CD44H is one mechanism used by CD14SUP+ peripheral blood monocytes to induce HA binding in response to inflammatory agents such as TNF-alpha and IFN-gamma.

12/7/13 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07745074 EMBASE No: 1999227441
Heparan sulfate composition of alternatively spliced CD44 fusion proteins
Piepkorn R.; Hovingh P.; Bennett K.L.; Linker A.
A. Linker, University of Washington, Box 356524, Seattle, WA United States
Biochemical and Biophysical Research Communications (BIOCHEM. BIOPHYS. RES. COMMUN.) (United States) 21 APR 1999, 257/3 (839-842)
CODEN: BBRCA ISSN: 0006-291X
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 20

Prior analyses of recombinant CD44 fusion proteins have indicated that combinatorial splicing of variant exons exerts distal effects on chondroitin sulfate content and structure, which may regulate the biological properties of the respective CD44 isoforms. The consequences of splicing of variant exons V4-7 on the heparan sulfate moieties were therefore examined, utilizing recombinant chimeras containing exons V3 and V8-10, engineered with or without exons V4-7 and expressed as Ig fusion proteins in COS cells. Splicing of exons V4-7, though they contain no

consensus motifs for glycosaminoglycan assembly, resulted in markedly increased polymer ***sulfation*** levels of the heparan sulfates. The sulfate groups of both the **CD44** V3-10 and V3,8-10 isoforms occurred as di- and tri-sulfated dissacharide units and were restricted to one N-sulfated block domain within the polymers. Compared to native human keratinocyte CD44, the recombinant heparan sulfates were relatively low in sulfate content. Our data indicate that variant exon V4-7 splicing exerts distal effects on the composition of this glycosaminoglycan. These effects may regulate those functions that are mediated through the heparan sulfate moieties, such as the binding of growth factors.

12/7/14 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11507609 PMID: 11673554
Role of **sulfation** in **CD44**-mediated hyaluronan binding induced by inflammatory mediators in human CD14(+) peripheral blood monocytes.
Brown K L; Maiti A; Johnson P
Department of Microbiology and Immunology, University of British Columbia, Vancouver, British Columbia, Canada.
Journal of immunology (Baltimore, Md. - 1950) (United States) Nov 1 2001, 167 (9) p5367-74, ISSN 0022-1767 Journal Code: 2985117R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Activation of T cells by Ag or stimulation of monocytes with inflammatory cytokines induces CD44 to bind to hyaluronan (HA), an adhesion event implicated in leukocyte-leukocyte, leukocyte-endothelial cell, and leukocyte-stromal cell interactions. We have previously shown that TNF-alpha induces **CD44 sulfation** in a leukemic cell line, which correlated with the induction of HA binding and **CD44**-mediated adhesion. In this study, we establish that TNF-alpha and IFN-gamma induce HA binding and the **sulfation** of **CD44** in CD14(+) PBMC, whereas no induced HA binding or **CD44 sulfation** was observed in CD14(-) PBMC stimulated with TNF-alpha. Treatment of cells with NaClO(3), an inhibitor of **sulfation** , prevented HA binding in a significant percentage of CD14(+) PBMC induced by TNF-alpha, LPS, IL-1beta, or IFN-gamma. Furthermore, stimulation with TNF-alpha or IFN-gamma in the presence of NaClO(3) reduced the ability of isolated CD44H to bind HA, demonstrating a direct effect of CD44H sulfation on HA binding. In contrast, the transient induction of HA binding in T cells by PHA was not affected by NaClO(3), suggesting that activated T cells do not use sulfation as a mechanism to regulate HA binding. Overall, these results demonstrate that inducible sulfation of CD44H is one mechanism used by CD14(+) peripheral blood monocytes to induce HA binding in response to inflammatory agents such as TNF-alpha and IFN-gamma.
Record Date Created: 20011023
Record Date Completed: 20011205

12/7/15 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

129188110 CA: 129(15)188110v JOURNAL
CD44 variant exon v5 encodes a tyrosine that is sulfated
AUTHOR(S): Sleeman, Jonathan P.; Rahmsdorf, Ursula; Steffen, Anja; Ponta, Helmut; Herrlich, Peter
LOCATION: Institute of Genetics, Forschungszentrum Karlsruhe, Karlsruhe, Germany,
JOURNAL: Eur. J. Biochem. DATE: 1998 VOLUME: 255 NUMBER: 1 PAGES:

74-80 CODEN: EJBCAI ISSN: 0014-2956 LANGUAGE: English PUBLISHER:
Springer-Verlag
SECTION:
CA215002 Immunochemistry
IDENTIFIERS: CD44 exon v5 tyrosine sulfated
DESCRIPTORS:
CD44(antigen)... Mouse... Rat... Sulfation...
CD44 variant exon v5 encodes sulfated tyrosine
Protein motifs...
tyrosine-sulfation; CD44 variant exon v5 encodes sulfated tyrosine
Exon(genetic element)...
v5; CD44 variant exon v5 encodes sulfated tyrosine
CAS REGISTRY NUMBERS:
956-46-7 CD44 variant exon v5 encodes sulfated tyrosine
?

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ds

Set     Items   Description
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S2          9   S1 AND (HECA(W)52 OF L(W)SELECTIN OR E(W)SELECTIN)
S3          5   RD S2 (unique items)
S4        158   (L(W)SELECTIN OR E(W)SELECTIN OR HECA(W)52)(20N)(SULFATION)
S5         63   RD S4 (unique items)
S6          7   S5(20N)(INDEPENDENT)
S7          7   RD S6 (unique items)
S8          7   HCELL
S9          4   RD S8 (unique items)
S10     15702   CD44
S11        40   S10(20N)(SULFATION)
S12        15   RD S11 (unique items)
? s (cd44)(20n)(glycoform?)
          15702  CD44
           2649  GLYCOFORM?
     S13     17  (CD44)(20N)(GLYCOFORM?)
? rd s13
...completed examining records
     S14      6  RD S13 (unique items)
? t s14/7/all

 14/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
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0013627075   BIOSIS NO.: 200200220586
Homing and hematopoiesis: HCELL is the principal E-selectin and L-selectin
  ligand of human hematopoietic stem cells
AUTHOR: Sackstein Robert (Reprint); Dimitroff Charles J (Reprint); Lee Jack
  Y (Reprint); Fuhlbrigge Robert C (Reprint); Parmar Kalindi; Mauch Peter M
  ; Sandmaier Brenda M
AUTHOR ADDRESS: Dermatology and Medicine, Brigham and Women's Hospital,
  Boston, MA, USA**USA
JOURNAL: Blood  98 (11 Part 1): p710a November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of
Hematology, Part 1  Orlando, Florida, USA  December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The selectins are becoming increasingly recognized for playing
  key roles in hematopoiesis. The endothelial selectins, E- and P-selectin,
  are both constitutively expressed on bone marrow (BM) microvascular
  endothelium, where they help mediate hematopoietic progenitor cell (HPC)
  migration into BM. Expression of the leukocyte selectin, L-selectin, on
  human CD34+ HPCs is associated with higher clonogenic activity in in
  vitro assays and faster engraftment following BM transplantation. Human
  HPCs also express PSGL-1, a ligand for all three selectins, however,
  paradoxically, engagement of PSGL-1 appears to inhibit clonogenic
  activity of human HPCs. These published data, collectively, have prompted
  us to explore the structure and distribution of selectin ligands
  expressed on human HPCs. Utilizing a new shear-based adhesion assay
  system developed in our laboratory, we have analyzed the cell surface
  glycoproteins of normal human HPCs that mediate L-, E- and P-selectin
  binding. Normal BM cells were separated into various lineage- and
  lineage+ subsets by magnetic bead sorting, and also sorted by flow
  cytometry of "side-population" cells following Hoechst dye staining. Cell
  membrane proteins were resolved into component bands by SDS-PAGE, then
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blotted onto PVDF. The blot was then placed in a flow chamber apparatus, and L-selectin+lymphocytes or stably transfected CHO cells bearing E- or P-selectin (designated CHO-E and CHO-P, respectively) were introduced into the chamber under controlled flow conditions. Adhesive interactions between cells in flow and immobilized (blot) proteins were visualized by video microscopy. CHO-P adhesive interactions occurred only at bands corresponding to PSGL-1. Adhesive interactions using lymphocytes and CHO-E were also observed at bands corresponding to PSGL-1, but significantly more L- and E-selectin ligand activity was observed at a band of apprx100,000 mw, operationally named "Hematopoietic Cell E-/L-selectin Ligand" (HCELL). Mass spectroscopy analysis of this protein, confirmed by immunopurification, revealed that this E- and L-selectin ligand is a previously unrecognized **glycoform** of a well-characterized glycoprotein, ***CD44*** . In shear-based adhesion assays of purified protein or of protein expressed naturally on cell membranes, HCELL displays >5-fold more avidity for E- and for L-selectin compared to PSGL-1. Though CD44 is broadly expressed among normal human BM marrow cells, HCELL is expressed only on lineage- cells: its expression is characteristic of CD34+ cells, with highest expression in CD38-/lin- cells. Additionally, HCELL is expressed on CD34+ and CD34- subsets of "side-population" cells. The distinctive, restricted expression of HCELL among the subsets comprising the human hematopoietic "stem" cell and its marked avidity for E- and L-selectin supports a role for this unique **glycoform** of **CD44** as a BM "homing receptor" as well as being the principal ligand mediating L-selectin-dependent cell-cell adhesive events within the BM microenvironment.

14/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013567681 BIOSIS NO.: 200200161192
Differential L-selectin binding activities of human hematopoietic cell L-selectin ligands, HCELL and PSGL-1
AUTHOR: Dimitroff Charles J; Lee Jack Y; Schor Kenneth S; Sandmaier Brenda M; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, Harvard Skin Disease Research Center, 77 Ave. Louis Pasteur, Boston, MA, 02115, USA**USA
JOURNAL: Journal of Biological Chemistry 276 (50): p47623-47631 December 14, 2001 2001
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Expression of L-selectin on human hematopoietic cells (HC) is associated with a higher proliferative activity and a more rapid engraftment after hematopoietic stem cell transplantation. Two L-selectin ligands are expressed on human HCs, P-selectin glycoprotein ligand-1 (PSGL-1) and a specialized **glycoform** of **CD44** (hematopoietic cell E- and L-selectin ligand, HCELL). Although the structural biochemistry of HCELL and PSGL-1 is well characterized, the relative capacity of these molecules to mediate L-selectin-dependent adhesion has not been explored. In this study, we examined under shear stress conditions L-selectin-dependent leukocyte adhesive interactions mediated by HCELL and PSGL-1, both as naturally expressed on human HC membranes and as purified molecules. By utilizing both Stamper-Woodruff and parallel-plate flow chamber assays, we found that HCELL displayed a 5-fold greater capacity to support L-selectin-dependent leukocyte adherence across a broad range of shear stresses compared with that of PSGL-1. Moreover, L-selectin-mediated leukocyte binding to immunopurified HCELL was consistently >5-fold higher than leukocyte binding to

equivalent amounts of PSGL-1. Taken together, these data indicate that HCELL is a more avid L-selectin ligand than PSGL-1 and may be the preferential mediator of L-selectin-dependent adhesive interactions among human HCs in the bone marrow.

14/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013153171 BIOSIS NO.: 200100325010
CD44 is a major E-selectin ligand on human hematopoietic progenitor cells
AUTHOR: Dimitroff Charles J; Lee Jack Y; Rafii Shahin; Fuhlbrigge Robert C; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Ave. Louis Pasteur, Room 671, Boston, MA, 02115, USA**USA
JOURNAL: Journal of Cell Biology 153 (6): p1277-1286 June 11, 2001 2001
MEDIUM: print
ISSN: 0021-9525
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: E-selectin plays a critical role in mediating tissue-specific homing of T cells into skin, and of primitive hematopoietic progenitor cells (HPCs) into bone marrow (BM). Though it is known that a glycoform of PSGL-1 (CLA) functions as the principal E-selectin ligand on human T lymphocytes, the E-selectin ligand(s) of human HPCs has not been identified. We used a shear-based adherence assay to analyze and define the E-selectin ligand activity of membrane proteins from human HPCs. Our data show that PSGL-1 expressed on human HPCs is an E-selectin ligand, and that HPCs also express a previously unrecognized E-selectin ligand, CD44. The E-selectin ligand activity of ***CD44*** is conferred by the elaboration of sialylated, fucosylated binding determinants on N-glycans. This **glycoform** of **CD44** is expressed on primitive CD34+ human HPCs, but not on more mature hematopoietic cells. Under physiologic flow conditions, this molecule mediates E-selectin-dependent rolling interactions over a wider shear range than that of PSGL-1, and promotes human HPC rolling interactions on E-selectin expressed on human BM endothelial cells. These findings offer new insights into the structural biology and physiology of CD44, and into the molecular basis of E-selectin-dependent adhesive interactions that direct homing of human HPC to BM.

14/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012896636 BIOSIS NO.: 200100068475
A distinct **glycoform** of **CD44** is an L-selectin ligand on human hematopoietic cells
AUTHOR: Dimitroff Charles J; Lee Jack Y; Fuhlbrigge Robert C; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, 77 Avenue Louis Pasteur, Room 671, Boston, MA, 02115, USA**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (25): p13841-13846 December 5, 2000 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We previously have obtained operational evidence of a hematopoietic cell L-selectin ligand expressed on normal human hematopoietic cells and on leukemic blasts. Using a technique developed in our laboratory for analyzing and identifying adhesion molecules, we show here that hematopoietic cell L-selectin ligand is a specialized ***glycoform*** of ***CD44*** . This L-selectin ligand activity of **CD44** requires sialofucosylated N-linked glycans and is sulfation-independent. These data provide important insights on the structural biology of CD44 and reveal a role for this protein as an L-selectin ligand on human hematopoietic cells.

14/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011321941 BIOSIS NO.: 199800116188
Glycosylation provides both stimulatory and inhibitory effects on cell surface and soluble CD44 binding to hyaluronan
AUTHOR: Skelton Timothy P; Zeng Chunxun; Nocks Aaron; Stamenkovic Ivan (Reprint)
AUTHOR ADDRESS: Dep. Pathol., Harvard Med. Sch. and Pathol. Res., Massachusetts General Hosp. East, 149 13th Street, Charlestown Navy Yard, Boston, MA 02129, USA**USA
JOURNAL: Journal of Cell Biology 140 (2): p431-446 Jan. 26, 1998 1998
MEDIUM: print
ISSN: 0021-9525
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Glycosylation has been implicated in the regulation of CD44-mediated cell binding of hyaluronan (HA). However, neither the relative contribution of N- and O-linked glycans nor the oligosaccharide structures that alter CD44 affinity for HA have been elucidated. To determine the effect of selective alteration of CD44 oligosaccharide composition on the affinity of CD44 for HA, we developed a novel strategy based on the use of affinity capillary electrophoresis (ACE). Soluble recombinant CD44-immunoglobulin fusion proteins were overproduced in the mutant CHO cell line ldl-D, which has reversible defects in both N- and O-linked oligosaccharide synthesis. Using this cell line, a panel of recombinant glycosidases, and metabolic glycosidase inhibitors, **CD44 glycoforms** with defined oligosaccharide structures were generated and tested for HA affinity by ACE. Because ldl-D cells express endogenous cell surface CD44, the effect of any given glycosylation change on the ability of cell surface and soluble CD44 to bind HA could be compared. Four distinct oligosaccharide structures were found to effect CD44-mediated HA binding: (a) the terminal alpha2,3-linked sialic acid on N-linked oligosaccharides inhibited binding; (b) the first N-linked N-acetylglucosamine residue enhanced binding; (c) O-linked glycans on N-deglycosylated CD44 enhanced binding; and (d) N-acetylgalactosamine incorporation into non-N-linked glycans augmented HA binding by cell surface CD44. The first three structures induced up to a 30-fold alteration in the intrinsic CD44 affinity for HA (Kd = 5 to >150 muM). The fourth augmented CD44-mediated cellular HA avidity without changing the intrinsic HA affinity of soluble CD44.

14/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011121818 BIOSIS NO.: 199799755878
Growth as a solid tumor or reduced glucose concentrations in culture

reversibly induce CD44-mediated hyaluronan recognition by Chinese hamster ovary cells
AUTHOR: Zheng Zhong; Cummings Richard D; Pummill Philip E; Kincade Paul W (Reprint)
AUTHOR ADDRESS: 825 N.E. 13th St., Oklahoma City, OK 73104, USA**USA
JOURNAL: Journal of Clinical Investigation 100 (5): p1217-1229 1997 1997
ISSN: 0021-9738
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The density, molecular isoform, and posttranslational modifications of CD44 can markedly influence growth and metastatic behavior of tumors. Many CD44 functions, including some involving tumors, have been attributed to its ability to recognize hyaluronan (HA). However, only certain CD44-bearing cells bind soluble or immobilized HA. We now show that CD44 made by wild-type Chinese hamster ovary (CHO-K1) cells and a ligand-binding subclone differ with respect to N-linked glycosylation. While both bear ***CD44*** with highly branched, complex-type **glycoforms**, **CD44** expressed by the wild type was more extensively sialylated. CHO-K1 cells which failed to recognize HA when grown in culture gained this ability when grown as a solid tumor and reverted to a non-HA-binding state when returned to culture. The ability of CHO-K1 cells to recognize HA was also reversibly induced when glucose concentrations in the medium were reduced. Glucose restriction influenced CD44-mediated HA binding by many but not all, of a series of murine tumors. Glucose concentrations and glycosylation inhibitors only partially influenced CD44 receptor function on resting murine B lymphocytes. These observations suggest that glucose levels or other local environmental conditions may markedly influence glycosylation pathways used by some tumor cells, resulting in dramatic alteration of CD44-mediated functions.
?

hematopoietic "stem" cell and its marked avidity for E- and L-selectin supports a role for this unique glycoform of CD44 as a BM "homing receptor" as well as being the principal ligand mediating L-selectin-dependent cell-cell adhesive events within the BM microenvironment.

----------sackstein ----------

9/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013567681 BIOSIS NO.: 200200161192
Differential L-selectin binding activities of human hematopoietic cell L-selectin ligands, HCELL and PSGL-1
AUTHOR: Dimitroff Charles J; Lee Jack Y; Schor Kenneth S; Sandmaier Brenda M; Sackstein Robert (Reprint)
AUTHOR ADDRESS: Harvard Institutes of Medicine, Harvard Skin Disease Research Center, 77 Ave. Louis Pasteur, Boston, MA, 02115, USA**USA
JOURNAL: Journal of Biological Chemistry 276 (50): p47623-47631 December 14, 2001 2001
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Expression of L-selectin on human hematopoietic cells (HC) is associated with a higher proliferative activity and a more rapid engraftment after hematopoietic stem cell transplantation. Two L-selectin ligands are expressed on human HCs, P-selectin glycoprotein ligand-1 (PSGL-1) and a specialized glycoform of CD44 (hematopoietic cell E- and L-selectin ligand, HCELL). Although the structural biochemistry of HCELL and PSGL-1 is well characterized, the relative capacity of these molecules to mediate L-selectin-dependent adhesion has not been explored. In this study, we examined under shear stress conditions L-selectin-dependent leukocyte adhesive interactions mediated by HCELL and PSGL-1, both as naturally expressed on human HC membranes and as purified molecules. By utilizing both Stamper-Woodruff and parallel-plate flow chamber assays, we found that HCELL displayed a 5-fold greater capacity to support L-selectin-dependent leukocyte adherence across a broad range of shear stresses compared with that of PSGL-1. Moreover, L-selectin-mediated leukocyte binding to immunopurified HCELL was consistently >5-fold higher than leukocyte binding to equivalent amounts of PSGL-1. Taken together, these data indicate that HCELL is a more avid L-selectin ligand than PSGL-1 and may be the preferential mediator of L-selectin-dependent adhesive interactions among human HCs in the bone marrow.

9/7/4 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

Query		Hit Count	Set Name result set
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L4	L3 same (sulfation)	2	L4
L3	('heca-452') same ('l-selectin' or cd62 or 'e-selectin')	7	L3
L2	L1 and ('heca-452' or sulfation)	4	L2
L1	sackstein.in.	11	L1

09/619290

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Search Results - Record(s) 1 through 2 of 2 returned.

☐ 1. Document ID: US 20030219836 A1

Using default format because multiple data bases are involved.

L4: Entry 1 of 2 File: PGPB Nov 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030219836
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030219836 A1

TITLE: Method of determining endometrial receptivity

PUBLICATION-DATE: November 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Fisher, Susan J.	San Francisco	CA	US	
Genbacev-Krtolica, Olga	Los Gatos	CA	US	
Prakobphol, Akraporn	Folsom	CA	US	
McMaster, Michael T.	Oakland	CA	US	

US-CL-CURRENT: 435/7.2; 530/388.1, 536/53

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

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☐ 2. Document ID: US 20030040607 A1

L4: Entry 2 of 2 File: PGPB Feb 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030040607
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030040607 A1

TITLE: Hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof

PUBLICATION-DATE: February 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Sackstein, Robert	Sudbury	MA	US	

US-CL-CURRENT: 530/395; 435/320.1, 435/325, 435/69.1, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs

Generate OACS

Search Results - Record(s) 1 through 7 of 7 returned.

☐ 1. Document ID: US 20030219836 A1

Using default format because multiple data bases are involved.

L3: Entry 1 of 7 File: PGPB Nov 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030219836
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030219836 A1

TITLE: Method of determining endometrial receptivity

PUBLICATION-DATE: November 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Fisher, Susan J.	San Francisco	CA	US	
Genbacev-Krtolica, Olga	Los Gatos	CA	US	
Prakobphol, Akraporn	Folsom	CA	US	
McMaster, Michael T.	Oakland	CA	US	

US-CL-CURRENT: 435/7.2; 530/388.1, 536/53

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

---

☐ 2. Document ID: US 20030148997 A1

L3: Entry 2 of 7 File: PGPB Aug 7, 2003

PGPUB-DOCUMENT-NUMBER: 20030148997
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030148997 A1

TITLE: Fluorinated glucosamine analogs useful for modulating post-translational glycosylations on cells

PUBLICATION-DATE: August 7, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Sackstein, Robert	Sudbury	MA	US	
Dimitroff, Charles J.	Brookline	MA	US	
Bernacki, Ralph J.	Elma	NY	US	
Sharma, Moheswar	Amherst	NY	US	
Matta, Khushi L.	Williamsville	NY	US	
Paul, Brajeswar	Williamsville	NY	US	

US-CL-CURRENT: 514/62; 514/161, 514/171, 514/406, 514/570, 536/53

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

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☐ 3. Document ID: US 20030086872 A1

L3: Entry 3 of 7 File: PGPB May 8, 2003

PGPUB-DOCUMENT-NUMBER: 20030086872
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030086872 A1

TITLE: SUT-2 and SUT-3 genes, proteins and assays for inhibitors of lymphocyte adhesion

PUBLICATION-DATE: May 8, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Girard, Jean-Philippe	Rebigue		FR	
Vincourt, Jean-Baptiste	Toulouse		FR	
Amalric, Francois	Toulouse		FR	

US-CL-CURRENT: 424/9.2; 435/183, 435/226, 435/320.1, 435/325, 435/69.1, 435/7.1, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

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☐ 4. Document ID: US 20030040607 A1

L3: Entry 4 of 7 File: PGPB Feb 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030040607
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030040607 A1

TITLE: Hematopoietic cell E-selection/L-selectin ligand polypeptides and methods of use thereof

PUBLICATION-DATE: February 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Sackstein, Robert	Sudbury	MA	US	

US-CL-CURRENT: 530/395; 435/320.1, 435/325, 435/69.1, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

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☐ 5. Document ID: US 20020019341 A1

L3: Entry 5 of 7 File: PGPB Feb 14, 2002

PGPUB-DOCUMENT-NUMBER: 20020019341
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020019341 A1

TITLE: Modulation of systemic memory T cell trafficking

PUBLICATION-DATE: February 14, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Butcher, Eugene C.	Portola Valley	CA	US	
Campbell, James J.	Palo Alto	CA	US	
Wu, Lijun	Reading	MA	US	
Rottman, James B.	Sudbury	MA	US	

US-CL-CURRENT: 514/8; 424/144.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

---

☐ 6. Document ID: US 6245332 B1

L3: Entry 6 of 7 File: USPT Jun 12, 2001

US-PAT-NO: 6245332
DOCUMENT-IDENTIFIER: US 6245332 B1

TITLE: Modulation of systemic memory T cell trafficking

DATE-ISSUED: June 12, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Butcher; Eugene C.	Portola Valley	CA		
Campbell; James J.	Palo Alto	CA		
Wu; Lijun	Reading	MA		
Rottman; James B.	Sudbury	MA		

US-CL-CURRENT: 424/184.1; 424/130.1, 424/139.1, 424/141.1, 424/145.1, 424/85.1, 514/1, 514/12, 514/2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. De

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☐ 7. Document ID: US 5962424 A

L3: Entry 7 of 7 File: USPT Oct 5, 1999

US-PAT-NO: 5962424
DOCUMENT-IDENTIFIER: US 5962424 A

TITLE: Methods and compositions for targeting selectins

DATE-ISSUED: October 5, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hallahan; Dennis E.	Park Ridge	IL		
Weichselbaum; Ralph R.	Chicago	IL		

US-CL-CURRENT: 514/44; 424/455, 424/458, 424/93.21, 536/24.1

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Term	Documents
HECA-452	22
HECA-452S	0
L-SELECTIN	1099
L-SELECTINS	68
CD62	249
CD62S	0
E-SELECTIN	1957
E-SELECTINS	59
(HECA-452 SAME (CD62 OR L-SELECTIN OR E-SELECTIN)).PGPB,USPT,EPAB,JPAB,DWPI.	7
(('HECA-452') SAME ('L-SELECTIN' OR CD62 OR 'E-SELECTIN')).PGPB,USPT,EPAB,JPAB,DWPI.	7

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